How Well Have We Answered the Arguments Against Regulating $PM_{2.5}$ in 1997?

Presenter: Joel Schwartz

Harvard University School of Public Health

research and development

Top 9 Reasons Not to Regulate

- Time series associations confounded
- Exposure uncorrelated with ambient
- It's all harvesting
- Thresholds
- No mechanism/biological plausibility
- Only due to some particles, will regulate wrong ones
- Don't know who is susceptible
- Only 2 cohort studies/faked
- Don't know if lower PM2.5 means fewer deaths

This poster will address the Epidemiologic Questions

Times Series Associations Confounded

- Case-Crossover/Matching
- Exposure Studies
- Hierachical Modeling Approach

Case Crossover Studies

- Match each person with themselves as a control on a nearby day when they did not die
  - --Bateson and Schwartz (1999,2001) showed how to choose so can control for Season
  - --Lumley (2000) showed how to choose to avoid Selection Bias
- Can Match on Same Concentration of Other Pollutant or Temperature to eliminate confounding
- 14 US Cities
- Controls Matched on Temperature
  - 0.39% (0.19—0.58) Increase per 10 mg/m^3 PM10 (Schwartz, OEM 2004)
- Controls Matched on Other Pollutants:
- CO 0.53% [0.04, 1.02]
- O_3 0.45% [0.12, 0.78]
- NO_2 0.78% [0.42, 1.15]
- SO_2 0.81% [0.47, 1.15]
  - Schwartz, EHP 2004
  - Two day mean gives larger effects
  - Not confounded

Exposure Issues

- Ambient pollution is a surrogate for personal exposure
  - Better measured pollutant will "steal" effect from worse measured pollutant
- Zeger et al (2000)
  - Stealing very unlikely
  - Bias is downward

Exposure Studies and Confounding

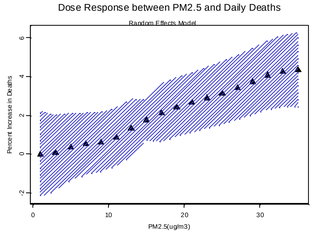
- In Baltimore and Boston
  - Ambient Ozone, NO2, SO2 are <u>better</u> predictors of Exposure to PM2.5 than of Exposure to themselves
  - NO2 and CO better predict traffic particles
  - Ozone better predicts Sulfates
- Suggests in Eastern US two pollutant models are just source apportionment for PM effects, and need personal monitoring to study gases

New Measurement Error Resistant Method

- Control for Confounding by Second Pollutant Across City in Meta-analysis
- Reduces Effect of Measurement Error (Schwartz and Coull, Biostatistics 2003)
- Example: Six City Study

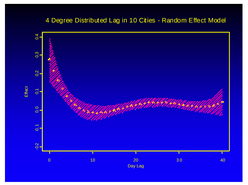
Particle Measure	Standard Estimate	Corrected Estimate
PM2.5	.0149 (.00197)	.0342 (.00287)
Coarse Mass	-.00206 (.00491)	-.0235 (.00616)

Threshold?



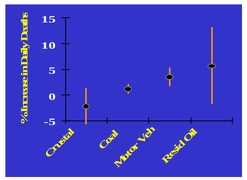
No threshold (confirmed by several studies)

Harvesting?



No harvesting (confirmed by several studies)

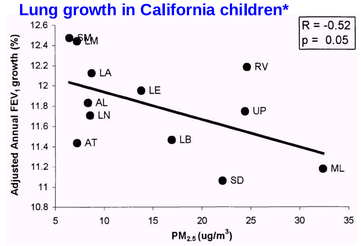
Only Some Sources Produce Toxic Particles



Sulfates, traffic particles, and residual oil all seem important

Epidemiology and Mechanisms

Lung growth in California children*



Mechanism: arterial dysfunction in diabetics

Associations between 6-day moving average exposure to particulate air pollutants and vascular reactivity, controlling for age, race, sex, BMI*, season, apparent temperature, and disease status (for total subjects estimate)

Subjects	Pollutant	n	Endothelium dependent % change per IQR † (95% CI ‡)	n	Endothelium independent % change per IQR (95% CI)
Type 2	Black carbon	148	-12.8 (-23.5, -0.6)	135	-6.8 (-15.1, 2.4)
	$PM_{2.5}$	183	-8.8 (-17.0, 0.1)	169	-8.5 (-14.1, -2.5)
	Particle #	125	-6.3 (-24.5, 16.2)	114	-11.1 (-23.8, 3.8)
	Sulfate	125	-12.1 (-19.3, -4.2)	115	-6.2 (-11.5, -0.6)

* Body mass index
† Interquartile range of the pollutant, for the days under consideration
‡ confidence interval

Sulfates and traffic both matter (O'Neill, in press)

Validity of Cohort Studies

- Cohort studies reanalyzed and found to be robust (HEI)

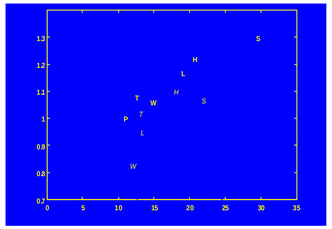
Research funded by others:

- New cohort in Netherlands finds effects of traffic particles on mortality
- Children's Health Study finds air pollutants (including particles) impair lung growth in children*

*Major funding provided by California Air Resources Board

If We Change Pollution, Does Mortality Change?

Follow-up of the Six City Study



New cohort study in the Netherlands shows even larger risks

Impact/Outcomes

- Epidemiology has proven to be a valuable tool to dissect human health outcomes associated with PM.
- Through several reanalyses and additional studies, the associations have proven robust and coherent.
- The application of statistical methods to diverse environments has provided distinctions between PM from varied sources.
- The epidemiology of PM has provided the core quantitative base for the risk assessments used in the development of the PM NAAQS.

Future Directions

Future epidemiology studies can address:

- Susceptibility – new groups that may be at increased risk from the effects of PM (developing fetus, diabetics)
- Mechanisms of toxicity
- Effects due to different sources/characteristics of particles
- Chronic effects

Health and Exposure